

GERIATRIC URETEROPELVIC JUNCTION OBSTRUCTION: THE POSSIBLE ROLE OF AN ARTERIOSCLEROTIC LOWER POLE BRANCH OF RENAL ARTERY: REPORT OF TWO CASES

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An 83-year-old woman presented with left flank pain and high grade fever. After left ureteral catheterization and intensive chemotherapy with hemoperfusion, surgical exploration revealed the lower pole branches of the renal vessels were obstructing the ureteropelvic junction (UPJ), and dissection of the vessels released the obstruction. An 82-year-old man presented with right flank pain. Angiography demonstrated UPJ obstruction caused by the lower pole branch of the renal artery. Arterial dissection with dismembered pyeloplasty resulted in improvement of obstruction. In both cases, the patients had a long history of hypertension with mild to severe arteriosclerosis.

Arteriosclerosis associated with fixation of the UPJ, may be one of the important factors leading to progressive hydronephrosis in geriatric patients.

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Key words: Ureteropelvic junction obstruction, Old age, Arteriosclerosis, Renal artery

INTRODUCTION

Ureteropelvic junction (UPJ) obstruction is the most common congenital anomaly seen in the urinary tract¹⁾. The exact cause of this anomaly remains an enigma but some conditions such as intrinsic stenosis, extrinsic mechanical compression by an external vessel or fibrous band and poor transmission of peristalsis, have been reported as possible causes¹⁾. Herein we describe 2 geriatric cases of UPJ obstruction, in which arteriosclerosis of a lower pole branch of renal artery might have played a pathogenic role.

CASE REPORTS

Case 1. An 83-year-old woman presented with complaint of a persistent, vague back pain on the left side of 3-month duration. The patient had a long history of hypertension. She was febrile (38°C) and her white blood cell (WBC) count was 15,000/mm³. C-reactive protein (CRP) was also abnormally elevated. Ultrasonography revealed left hydronephrosis. Retrograde ureteral stenting yielded much purulent fluid. A plain computed tomography (CT) scan revealed no apparent abnormalities in size and configuration of the left renal parenchyma. Since the serum endotoxin level was 13.8 pg/ml (reference range <10 pg/ml), intensive chemotherapy with hemoperfusion using a polymyxin B affinity column was started. CRP returned to the reference range within 3 weeks. Since the renogram showed functional deterioration of the left kidney, surgical exploration was performed 2 months later. The

lower pole branches of the renal artery and vein were severely sclerotic with fibrous adhesion to UPJ, and

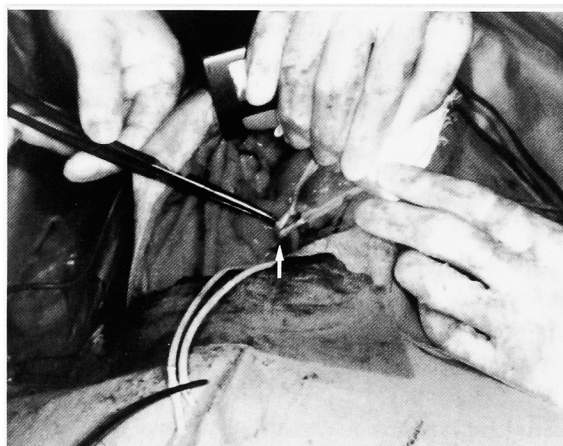
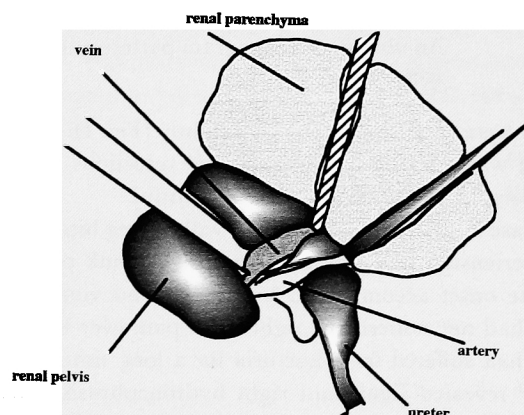


Fig. 1. The ureteropelvic junction was compressed by lower pole branches of renal artery and vein (arrow). A diagram is provided for clarity.

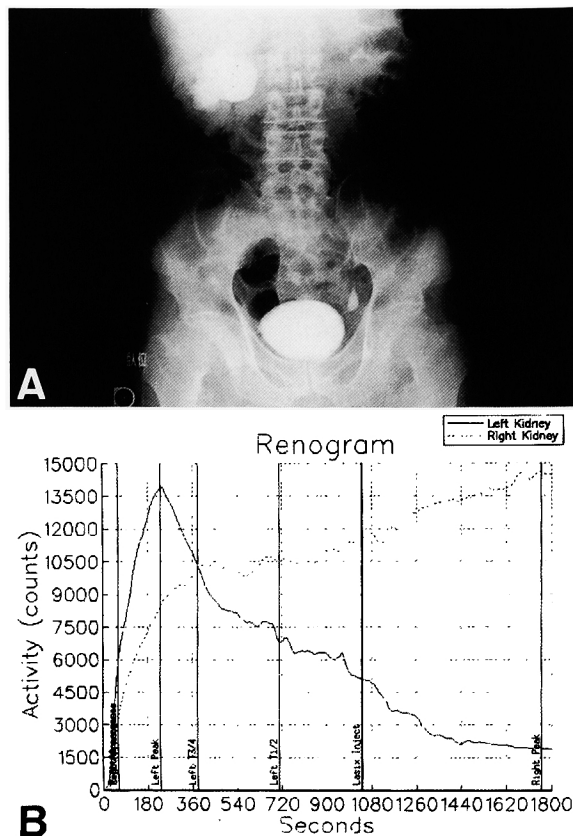


Fig. 2. A: IVP revealing right hydronephrosis associated with UPJ obstruction. B: An obstructive renogram pattern can be seen.

they were compressing the UPJ tightly (Fig. 1). The UPJ was released by ligations and resections of the vessels. Convalescence was uneventful.

Case 2. An 82-year-old man with a long history of hypertension was seen for severe right flank pain of acute onset accompanied by nausea and vomiting. He had not noticed the right flank pain ever before. He had suffered from nocturia for a long time. An IVP revealed significant right hydronephrosis (Fig. 2A). Diuretic renogram washout patterns indicated obstruction of the UPJ (Fig. 2B). An abdominal CT scan and angiography demonstrated UPJ obstruction caused by the lower pole branch of the renal artery (Fig. 3A, 3B). Open laparotomy was performed on August 1996. The UPJ was angulated by the mild sclerotic artery with fibrous adhesion and also showed intrinsic ureteropelvic obstruction. Arterial dissection and Anderson-Hynes pyeloplasty was performed. The patient has remained free of symptoms ever since.

DISCUSSION

Surgical exploration revealed fixation of the UPJ by a lower pole branch of the renal artery in both cases. The renal pelvis protruded anteriorly over a lower pole branch of the renal artery. The angulation of the UPJ and hooking of the ureter interfered with

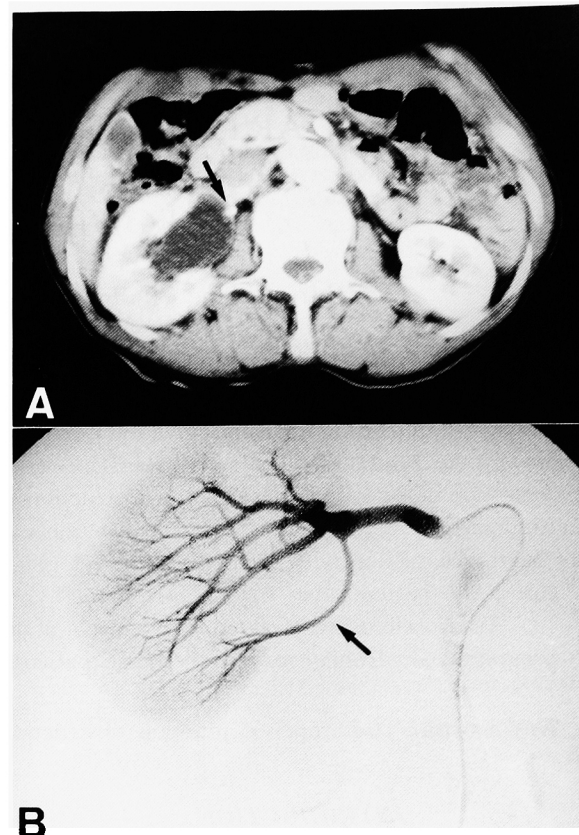


Fig. 3. A: CT scan revealing a dilated renal pelvis in relation to a lower pole branch of renal artery (arrow). B: Angiography showing a lower pole branch of renal artery (arrow).

urinary transport.

A long history of hypertension and medication with antihypertensive agents suggested that hardening and angulation of the UPJ promoted by arteriosclerosis of a lower pole branch of the renal artery triggered this progressive obstruction.

As the UPJ angulates, compression at the site where the fixed vessels cross it, increases. If the artery were elastic, without arteriosclerosis, the angulation and hooking might be set free. This is the reason why a UPJ obstruction is not diagnosed until the patient is in his/her eighties.

In case 1, during the 3 months of obstruction the helical muscle responsible for peristaltic transport²⁾ gradually elongated due to periodical overstretching³⁾. The elongation of this muscle might have promoted the obstruction. Incidental retrograde urinary infection might have fostered collagen deposition at the UPJ and the UPJ gets "sick"⁴⁾. This would promote the progressive UPJ obstruction, followed by septicemia.

Diuresis is another causative factor of UPJ obstruction^{5,6)}. In case 2, a long history of nocturia associated with dihydropyridine calcium channel blocker-induced excess urine output might have played a role in its pathogenesis. Incidental

compression of the UPJ due to arteriosclerosis might have reduced urinary transport, and urinary excretion exceeded the marginal UPJ function. In this case, since excision of the involved vessel did not relieve the obstruction, intrinsic UPJ hypofunction might also have played a role in its pathogenesis.

Hydronephrosis due to UPJ obstruction progressed rapidly in both cases but the course of deterioration of renal function differed between the two cases⁷⁾. Although we had no information regarding the renal function before presentation, a rapid deterioration was suspected in case 1 by CT scan and renogram, which revealed discrepancies between size and function of right and left kidney. In case 2, the renogram revealed normal renal function.

Although the exact reason for this difference is unknown, urinary tract infection and difference in initial renal function might have been involved.

Ureteropelvic junction obstruction is usually thought to be congenital. However, acquired obstruction in adults, which may rapidly progress without adequate warning, is less commonly seen^{7,8)}. Recently Yoshida et al. reported that arteriosclerotic change of an upper pole branch of the renal artery was the cause of hydronephrosis⁹⁾. In conclusion, arteriosclerosis of a lower pole branch of renal artery might be an etiological factor of geriatric UPJ obstruction.

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動脈硬化を伴う腎下極への腎動脈分枝による高齢者の
腎盂尿管移行部狭窄症の2例

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症例1は83歳，女性．主訴は左側腹部痛と高熱．左尿管カテーテル留置し，抗生剤投与とエンドトキシン吸着療法施行後，手術にて腎下極を支配する動静脈による腎盂尿管移行部の圧迫が左水腎症の原因であると判明．これらの切断にて閉塞を解除した．症例2は82歳，男性．主訴は右側腹部痛で，既往歴に高血圧があった．腎血管造影にて腎下極への動脈の圧迫による

水腎症が原因と判明．この動脈の切断と腎盂形成術にて軽快．2例とも長期に高血圧症を患い，軽度から重度の動脈硬化を呈していた．

動脈硬化による腎盂尿管移行部の締め付けが，高齢者に発症し，増悪する水腎症の一因と考えられる．

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